Methods: Intravascular ultrasound (IVUS), fractional flow reserve (FFR), index of microcirculatory resistance (IMR) and blood examination (CK-MB, cardiac troponin (cTn) were performed in 82 consecutive patients with stable angina pectoris undergoing PCI. FFR and IMR were measured with a single coronary pressure wire. IMR was defined as Pd/coronary flow (or Pd * mean transit time) at peak hyperemia. Patients were randomized to control (n=40) or nicorandil groups (n=42). In the nicorandil group, nicorandil was intravenously administered as a 6-mg bolus injection just before PCI and as a constant infusion at 6-mg/hour for 24-hours thereafter.
Results: All volumetric IVUS parameters and FFR were similar between the 2 groups both pre and post PCI. However, patients treated with nicorandil had significantly lower IMR immediately post PCI, and tended to have lower cTn 24-hours post PCI (IMR: 25.4±12.1 vs 17.9±9.1 units, p<0.05, and cTn:0.21±0.13 vs 0.12±0.08 ng/mL, p=0.06, for control vs. nicorandil).Incidence of cTn elevation more than 5-fold of normal range (<0.15ng/mL) was significantly larger in the control group than in the nicorandil group(41% vs. 12%, p<0.01). The control group showed a closer correlation between plaque volume reduction by IVUS and IMR than the nicorandil group(r=0.48 vs. 0.40, p<0.001 for control vs. nicorandil).

	Control	Nicorandil	p-value
Pre-Intervention			
Lumen (mm2)	4.12 ± 1.35	4.26 ± 1.49	0.45
Vessel (mm2)	13.8 ± 3.18	13.87 ± 3.91	0.93
Plaque (mm2)	9.67 ± 2.91	9.68 ± 3.01	0.87
FFR	0.70 ± 0.09	0.71 ± 0.09	0.84
Post-Stent			
Lumen (mm2)	8.18 ± 1.75	8.22 ± 2.02	0.92
Vessel (mm2)	16.73 ± 3.32	16.7 ± 4.11	0.97
Plaque (mm2)	8.58 ± 2.23	8.57 ± 2.71	0.99
FFR	0.93 ± 0.1	0.91 ± 0.10	0.53

Conclusions: In patients undergoing successful coronary stenting for stable angina, administration of nicorandil is associated with reduced microvascular dysfunction induced by PCI.

TCT-712

Can Amelioration of HDL-C Level by Pitavastatin Improve Clinical Outcomes in Acute Myocardial Infarction Patients Undergoing Percutaneous Coronary Intervention?

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Background: High density lipoprotein (HDL)-cholesterol may play an important role in improving prognosis by cardio-protective effect. However, there are limited data whether the improvement of HDL-C level by pitavastatin (Livalo®) in acute myocardial infarction (AMI) patients (pts) can reduce cardiovascular events or not.
Methods: A total of 404 consecutive AMI pts underwent percutaneous coronary intervention (PCI) with drug eluting stents (DES) from 10 major centers were divided into two groups; the improved HDL-C group (n=162) and the Control group (no improvement in HDL-C level, n=242). All pts were exclusively treated with pitavastatin 2~4mg/day from the onset of AMI. Improved HDL-C group was defined as increased HDL-C/Total Cholesterol ratio and/or increased HDL-C level from index to 6 months laboratory follow up. Major clinical outcomes up to 12 months were compared between the two groups.
Results: Baseline clinical characteristics were similar between the two groups except the improved HDL-C group showed higher level of triglyceride (146.8 vs. 111.8mg/dl, p<0.001) and lower HDL (39.7 vs. 50.3mg/dl, p<0.001). Procedural success rate and in hospital clinical outcomes were similar between the two groups. At 12 months, major clinical outcomes were not different between the two groups (table).
Conclusions: In AMI pts undergoing PCI with DESs exclusively treated by pitavastatin, improvements in HDL-C Level was not associated with better clinical outcomes.

Table: HDL-C Level Change and 12-month Clinical Outcomes

Variables, n (%)	Improved HDL-C (n=156 pts)	Control (n=233 pts)	P-value
HDL level			
On Admission	39.7±9.6	50.3±34.4	<0.001
1Month	44.7±10.3	44.7±44.7	0.990
6Month	48.4±11.2	41.5±10.2	<0.001
12Month	44.3±9.5	42.9±9.9	0.404
Cumulative 12-month clinical outcomes			
Total Death	1 (0.6)	1 (0.4)	0.775
Cardiac Death	1 (0.6)	1 (0.4)	0.775
Recurrent MI	1 (0.6)	5 (2.1)	0.238
Q-MI	0 (0.0)	3 (1.3)	0.155
Revascularization	17 (10.9)	30 (12.9)	0.557
TLR	13 (8.3)	12 (5.2)	0.210
TVR	14 (9.0)	23 (9.9)	0.768
TLR MACE	14 (9.0)	15 (6.4)	0.350
TVR MACE	16 (10.4)	26 (11.3)	0.778

TCT-713

The benefit of beta-blocker therapy in hospital survivors receiving primary percutaneous coronary intervention after ST-elevation myocardial infarction from the Korea Acute Myocardial Infarction Registry

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Background: The efficacy of beta-blockers (BBs) therapy in ST-elevation myocardial infarction (STEMI) patients who underwent primary percutaneous coronary intervention (PCI) is controversial. Moreover, the benefit of BBs therapy in hospital survivors who underwent primary PCI has not been fully investigated.
Methods: Between November 2005 and January 2008, 2,688 hospital survivors who had an STEMI with a symptom-to-door time of 12 hours and underwent primary PCI were analyzed from the Korean Acute MI registry. Patients who received BBs therapy before hospitalization were excluded from this study. The 12-month MACE was defined as a composite of death, non-fatal MI, and revascularizations.
Results: Of these patients, BBs were used in 2,042 (76.0%) hospital survivors. Patients receiving BBs were younger with less dyspnea at presentaion, higher body mass index, longer symptom-to-door time, higher systolic and diastolic blood pressure, higher left ventricular ejection fraction, and higher serum levels of glucose, total cholesterol, and triglyceride. Ventricular arrhythmia during hospitalization was less frequently observed in BBs patients. In Cox proportional-hazards model, there was no significant difference in the 12-month MACE between BBs patients and no-BBs patients (9.9% versus 11.1%; crude hazard ratio [HR] 0.864, 95% confidence interval [CI] 0.660–1.131; p=0.287). The 12-month mortality was significantly lower in BBs patients compared with no-BBs patients (2.0% versus 3.9%; crude HR 0.498, 95% CI 0.302–0.820; p=0.006). Propensity scores (PS) for BBs use was calculated for each of the patients, and was used to match 604 patients not receiving BBs with 604 patients receiving BBs. In Cox proportional-hazards model, there were no significant differences in the rate of 12-month MACE (10.6% versus 10.3%; HR 1.030, 95%CI 0.726–1.460; p=0.869) and mortality (2.8% versus 3.3%; HR 0.850, 95%CI 0.445–1.622; p=0.621) between BBs and no-BBs patients.
Conclusions: The benefit of BBs therapy might be less cardioprotective in hospital survivors with STEMI who underwent primary PCI. Further studies are required in these patients.

TCT-714

A Meta-Analysis of Randomized Controlled Trials Appraising The Efficacy and Safety of New Oral Antithrombotics in Patients with Acute Coronary Syndrome

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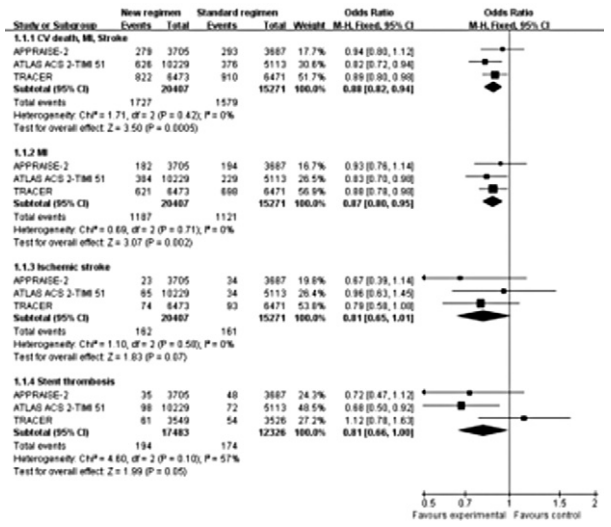
Background: Recently introduced oral antithrombotics, including apixaban, rivaroxaban, and vorapaxar, has been developed as alternative to conventional antiplatelet regimen for patients with acute coronary syndrome (ACS).
Methods: We searched MEDLINE, EMBASE, and Cochrane databases for randomized controlled trials (RCTs) comparing the efficacy and safety of new oral antithrombotics to standard antiplatelet regimen in patients with acute coronary syndrome. The primary efficacy end point was the composite of cardiovascular death, myocardial infarction, or stroke. Fixed-effects or Random-effects models were used to pool efficacy and safety data across RCTs.
Results: Three studies, including 35,862 patients, were identified. Patients randomized to a new oral antithrombotics had a decreased risk of the composite end point of cardiovascular death, MI, or stroke (odds ratio [OR], 0.88; 95% confidence interval [CI], 0.82 to 0.94) and myocardial infarction (OR, 0.88; 95% CI, 0.80 to 0.95). Incidence of ischemic stroke (OR, 0.81; 95% CI, 0.65 to 1.01) and stent thrombosis (OR, 0.81; 95% CI, 0.66 to 1.00) tended to be lower in patients randomized to a new oral antithrombotic

regimen. Randomization to a new oral antithrombotics was associated with a significantly increased risk of major bleeding (OR, 1.99; 95% CI, 1.65 to 2.39).



Conclusions: In patients with ACS, the addition of new oral antithrombotic agent to standard antiplatelet therapy significantly reduced the primary composite end point but significantly increased the risk of major bleeding.

TCT-715

First Demonstration of dose Dependent Effect of Statin Therapy on Urinary 11-dehydrothromboxane B2 levels in Patients Undergoing Coronary Angiography

Anand Singla[1], Kevin Bliden[2], Peter Toth[3], Udaya Tantry[2], Young-Hoon Jeong[4], Ali Tabrizchi[2], Denny Mathew[2], Gordon Ens[5], Paul Gurbel[2]
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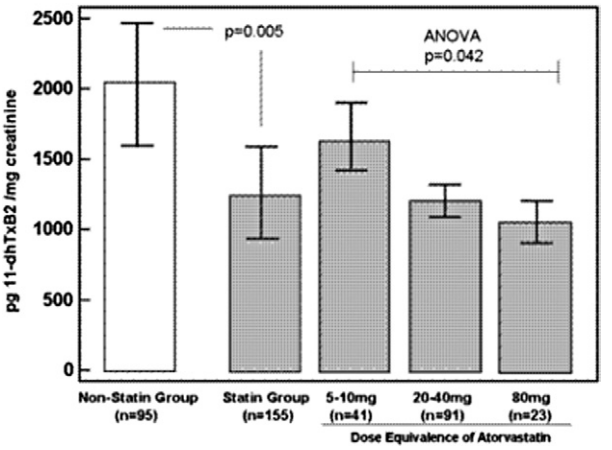
Background: Aspirin and statin therapy are primary treatment strategies in patients with cardiovascular disease. Heightened urinary 11-dehydrothromboxane B2(11-dTxB2) levels have been associated with an increased risk of adverse cardiovascular events. The aim of the study was to determine the effect of statin therapy on 11-dTxB2 levels in patients undergoing coronary angiography on 325 mg aspirin.
Methods: Urinary 11-dTxB2 was measured in patients prior to coronary angiography with (n=155) and without (n=95) statin therapy. Lipoproteins by vertical density gradient ultracentrifugation technique and thrombin-induced platelet-fibrin clot strength(TIP-FCS), a marker for cardiovascular events by thrombelastography, were also measured. The dose dependent effect of statin therapy was analyzed according to atorvastatin equivalent dose, categorized as: 5-10mg, 20-40mg, 80mg.
Results: Baseline demographics and cardiac risk factors were similar between groups. Statins significantly reduced urinary 11-dTxB2 levels in a dose dependent manner (Figure). There was a significant relation between quartiles of 11-dTxB2 and TIP-FCS,(p=0.05 for ANOVA). Patients on statin therapy had significantly lower levels of LDL, triglycerides, VLDL, atherox and apo B100 (p<0.005 for all). However, Urinary 11-dTxB2 did not correlate with lipids and lipoproteins measurements.
Conclusions: This is the first study to demonstrate the dose dependent response of statin therapy on urinary 11-dTxB2 levels, independent of lipid lowering effects.Clinical outcome studies are required to determine the utility of this novel marker for optimizing statin therapy.



TCT-716

First Simultaneous Assessment of the Effects of Fish Oil Supplementation on Lipid and Thrombogenicity Profiles

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Background: Fish Oil supplementation may have anti-atherosclerotic effects by reducing triglycerides, improving endothelial function, and reducing inflammation. However, inconsistent results have been reported on the effects of fish oil on platelet function. We aimed to determine if fish oil supplementation reduced the atherothrombotic risk profile by measuring lipids and thrombogenic factors simultaneously.
Methods: Patients with suspected coronary artery disease with (n=79) and without (n=324) fish oil supplementation were enrolled in the Multi-Analyte, Thrombogenic, and Genetic Markers of Atherosclerosis (MAGMA) study. The lipid profile was determined by vertical density gradient ultracentrifugation technique, and thrombogenicity by thrombelastography immediately prior to elective coronary angiography. Urinary 11-dehydrothromboxane B2 and AtherOx were performed by immunoassay. ADP and collagen-induced platelet aggregation were measured in aspirin+ clopidogrel- treated patients.
Results: Baseline demographics and cardiac risk factors were similar between groups. Fish oil supplementation was associated with significantly lower triglycerides, VLDL, remnant lipoproteins, and AtherOx levels and increased HDL levels (p<0.05). Among patients on aspirin + clopidogrel therapy, fish oil supplementation was associated with lower collagen-induced platelet aggregation (p=0.05, Table).

	No Fish Oil (n=324)	Fish Oil (n=79)	
	Mean ± SD	Mean± SD	p-value
Lipid Profile (mg/dL)			
Total cholesterol	161±38	162±52	0.85
Total low density lipoprotein	93±30	94±43	0.87
Total high density lipoprotein	44±12	49±17	0.03
Total very low density lipoprotein	22±13	19±6	0.02
Triglycerides	126±90	103±57	<0.05
Remnant lipoproteins	24.3±11	21.0±7.6	0.02
Thrombogenicity Profile			
AtherOx (mg/dL)	0.42±0.48	0.29±0.27	<0.05
Lipoprotein (a) (mg/dL)	8.6±6.3	8.4±6.0	0.82
11-dehydrothromboxane B2 (pg/mg creatinine)	1633±1636	1330±1520	0.25
Clotting index	−0.7±2.6	−0.5±3.2	0.12
MAKH (mm)	66.6±6.1	65.5±4.5	0.21
R (min)	7.9±2.3	7.4±2.9	0.12
20uM ADP-induced aggregation	52±20	46±21	0.2
4ug/mL collagen-induced aggregation	48±22	38±22	0.05